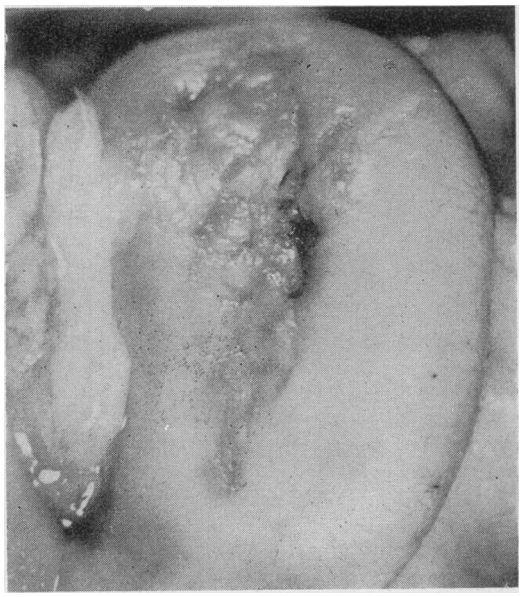

Fig 3 *Proliferation lesions on medial condyle of femur*

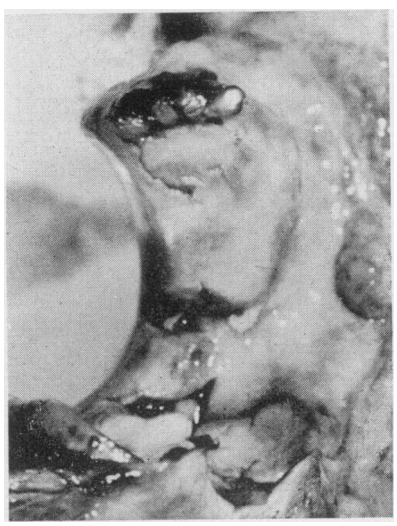

Fig 4 *Lift lesions in the ulnar notch*

The ætiology of these lesions most probably lies in the rapid growth rate and carcase characteristics that have been two of the three goals in modern pig breeding. It can be shown that bone growth in a rapidly growing pig is unable to keep pace with the growth in body weight. A similar phenomenon has been observed with respect to muscle fibre diameter (Staun 1963). Thus the rapidly grown pig of, say, 100 kg may have had less bone and thinner muscle fibres than a litter mate that has taken more time to reach the same weight. The small amount of exercise allowed to the intensively reared pig further limits muscle hypertrophy.

Schilling (1963) has made a detailed study of the effects of breeding pigs with longer backs and a greater mass of muscle on the inner aspect of the thigh. In his opinion, changes in the angles of attachment of muscles and changes in the angles of joints to accommodate greater masses of muscle contribute to the production of arthropathy.

It seems, therefore, that a number of factors predispose to joint lesions. Compared with its ancestor of twenty years ago, the modern pig has a greater body weight for its age and more muscle, although the latter may be rather weak and attached at unnatural angles to immaturely sized bones aligned in a suboptimal way. In the more cleanly conditions of some modern pig buildings, the floor is also often slippery. Under these conditions a high incidence of arthritic lesions becomes understandable.

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Mr M D N Hunt (*Newmarket*)

Traumatic Arthritis in Young Thoroughbreds

Clinically, traumatic arthritis differs fundamentally from the degenerative arthritis recorded in the mature horse and described under various names in the literature, in that it appears in the immature animal (Hare 1927, Mitchell 1937, Kelser & Callender 1938). It is considered by many to be essentially a second category of degenerative arthritis (osteoarthritis) differing from the disease of the mature animal only in its age incidence, its more rapid development and its predilection for certain joints. However, as it is almost certainly due to trauma that the first clinical signs become apparent, and as the full pathological picture is not yet established, whereas that of the older animal is essentially a non-inflammatory, degenerative disease, I prefer at the present to retain the term traumatic arthritis.

Incidence

The horse may be said to reach full skeletal maturity at about 4½ years. This disease is usually first recognized clinically in the yearling and 2-year-old thoroughbred, although occasionally it

may appear in even younger animals. Unlike all other breeds, the potential racehorse often begins light training by the time it is 18 months old and may have been raced before it is 2 years old.

The joints most commonly affected are the radiocarpal and intercarpal articulations of the carpus, the metacarpophalangeal (fetlock) joint and the coronopedal (coffin) joint. The disease is usually localized to one or two joints, and may be unilateral or bilateral. The proximal joints of the forelimbs and the joints of the hindlimbs are very rarely involved in the young thoroughbred; this is in direct contrast to the disease encountered in the older and coarser-bred animal where the tibiotarsal and intertarsal joints of the hindlimb are frequently the sites of osteoarthritis.

Signs

The first clinical sign of the disease is usually a slight distension of the joint capsule. With continued work many horses soon lose their fluency of gait or become slightly lame. It is at this time that the veterinarian may be consulted, but many trainers seem resigned to the fact that a percentage of their yearlings and 2-year-olds will 'lose their action' and become less impressive movers during the period following breaking. Radiological examination of the affected joint often reveals advanced pathological changes in the shape of osteophytic outgrowths on the articular borders. In a few cases, small fractured portions of these outgrowths are present as 'joint mice'. In those cases where the carpal joints are involved, in addition to the articular lesions, there may be periostitis and exostoses on the dorsal surfaces of the carpal bones at the sites of insertion of the interosseus ligaments. Early in the disease, the incidence of osteophytic formation varies considerably from joint to joint, being most common in the carpal joints. Continuation of exercise results in a clinical deterioration of the condition with excessive degree of new bone formation associated with the joint; an increasing incidence of joint mice; and in some cases calcification in the collateral ligaments or joint capsule where these structures attach to the periosteum. A decrease in the radiolucent joint space has been described but I believe that this is more characteristic of osteoarthritis (degenerative arthritis) of the intertarsal joints seen in older subjects. Analysis of fluid from a considerable number of joints of young thoroughbreds affected with traumatic arthritis shows them to correspond fairly closely to Group I fluids in the classification in humans (Bauer *et al.* 1940).

Pathology

It is in this fundamental aspect of the disease that least is known. The opportunities of studying the sequence of pathological changes in this condition are few and far between. Often by the time an affected animal comes to autopsy it is well past the age of maturity and lesions of degenerative arthritis are also present, causing considerable difficulty in interpretation.

Mackay-Smith (1962) states that the gross and microscopic changes seen in the degenerative arthritis of mature horses, first described in detail by Kelser & Callender (1938), and the osteoarthritis of the young thoroughbred – which I am calling traumatic arthritis – are similar, but that in the latter they develop more rapidly and the classical early stages may not be clearly discernible. In true degenerative or osteoarthritis of the adult the first changes appear in the cartilage. It is not until the subchondral surfaces are exposed and eburnation becomes marked that periarticular proliferation is seen (Sippel 1942). Primarily these proliferations are formed of cartilage. Bone then extends into these processes from the region of the calcified matrix and eventually the outgrowth is composed of cartilage-covered bone. We know that erosion of articular cartilage in itself is probably painless and that therefore it is difficult to estimate the time of commencement of the first changes within the joint. However, the early radiological recognition of osseous proliferations in the condition affecting young horses allows us to question whether the pathological pattern of these two types of osteoarthritis are indeed identical.

It appears that changes in the joint capsule are not generally very marked, although there is usually some inflammation and proliferation of the synovial membrane characterized by its thickness and by enlargement of the villi. This is especially common close to the marginal zone between articular cartilage and synovial membrane.

Ætiology

Trauma: It is usually accepted that the immediate exciting cause is trauma. This is mild, continual use-trauma produced initially by the stress imposed upon the animal during breaking in and later by training and racing. A horse of 2 or 3 years old is under physical strains unknown to a child of comparable age. A 1,000 lb racehorse carrying an 8 stone jockey over three-eighths of a mile in thirty-four seconds, generates a kinetic energy of over 1,850,000 ft lbf (Jenny 1960). This tremendous thrust is absorbed at one phase of the gallop on a single foreleg. The stress imposed on the anterior peripheral regions of the articular surfaces of the carpal bones is very evident in action photographs in which the degree of hyperextension of the carpus may readily be appreciated. The most commonly affected bone, the

radial carpal (scaphoid), is subjected to particularly severe compression forces, and in addition to arthritic changes this bone is commonly the seat of stress fractures, pathological specimens almost invariably showing severe bruising of the soft tissues within the bone (Rooney 1963). Although it is widely assumed that the site of lesions in a joint is the one exposed to the greatest stress of weight-bearing it has been suggested that injury to the cartilage by the disturbance of fluid permeation and hence nutrition is a more important factor than the mechanical stress (Harrison *et al.* 1953).

Posture: It has long been known that abnormal skeletal stresses arising from postural and structural abnormalities can induce pathological changes in young, healthy cartilage. Thoroughbreds are particularly prone to poor conformation which is often inherited. Whether the inheritable factor regarding the susceptibility of the thoroughbred to arthritis is purely one of conformation or whether there is a further genetical basis similar to the 'arthritis-prone' and 'arthritis-resistant' strains of mice reported by Sokoloff poses an interesting question (Sokoloff *et al.* 1960).

Nutrition: Unknown dietary deficiencies (Mitchell 1937, Kelser & Callender 1938), vitamin A deficiency (Howell *et al.* 1941), and trace element deficiencies have all been reported as the cause – direct or indirect – of arthritis. However, it is the role of calcium and phosphorus metabolism in joint disease that has received the most attention since it was found that either a deficiency of calcium or an imbalance in the calcium/phosphorus ratio could cause osteomalacia with accompanying joint lesions in the horse (Kitner & Holt 1932). However, the histopathology of equine osteomalacia is that of osteitis fibrosa and quite different from that of degenerative or inflammatory osteoarthritis. Furthermore, in recent work on the condition in which the bone changes were shown to be due to secondary hyperparathyroidism, no joint lesions were observed (Krook & Lowe 1964). However, many of the clinical features characteristic of osteitis fibrosa in the horse, such as stress fractures and the tearing of ligaments at their point of attachment to bone, are commonly seen accompanying traumatic arthritis in the racehorse. It is also true that young thoroughbreds often receive rations that are low in calcium and with a calcium/phosphorus imbalance. Since the last war, the feeding of higher protein diets to weanlings and yearlings together with the sometimes overzealous use of proprietary 'shot-gun' mineral-vitamin supplements has become popular and appears to have coincided with an increased incidence of enchondroses such as epiphysitis as well as arthritis.

Conclusion

Our knowledge of traumatic arthritis in the young thoroughbred is still superficial. Important though the effects of wear, tear and injury undoubtedly are, much deeper underlying factors are probably involved in the production of this disease.

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Dr W S C Copeman (*London*) said that before the last war he had been provided with facilities for studying every case of arthritis which occurred at the London Zoo. Osteoarthritis was commonly found in the heavy wild animals which were limited in their cage space, especially bears. It had been noted that the cartilage lesions were not always situated at the areas of maximum pressure, and that cartilage everywhere appeared to show a limited capacity for repair even in old animals. It had also been noted, as the result of X-ray studies, that the trabecular architecture of the weight-carrying bones was of importance in determining certain lesions. In some cases local blood-vessel infarction seemed to initiate aseptic necrosis or degeneration within the region supplied by it.

He suggested that in investigating the viscosity of joint fluid insufficient attention had been paid to hyaluronic acid metabolism, and mentioned that in the field of human osteoarthrosis the belief was growing that the basic defect might lie in a diminished capacity of the chondrocytes to produce sulphated mucopolysaccharides, thus leading to excessive loss of chondroitin sulphate.

Dr Copeman felt that it was difficult to see from the published experiments how the effect of the various procedures on animal arthrosis could constitute other than relatively nonspecific influences upon growth and metabolism. This would tie up with the failure to demonstrate in human cases any clear relationship between endocrine or chemical abnormalities and the progress of the disease.

Speaking of rheumatoid arthritis he stressed the need for a comparable experimental form in animals if further progress in knowledge was to be achieved. He pointed out that at present arthritis produced in animals was not self-perpetuating.